Cell Biology

A RISE IN INTRACELLULAR CALCIUM STIMULATES VOLUME DECREASE IN *NECTURUS* ERYTHROCYTES. Corryn Siegel and Douglas B. Light*. Department of Biology, Ripon College, Ripon, WI 54971 (siegelc@ripon.edu and lightd@ripon.edu).

The ability of animal cells to regulate volume is a fundamental property common to a large number of cell types and is evolutionarily one of the oldest regulatory mechanisms. Volume regulation is of importance in cells exposed to anisotonic extracellular media and in cells where transport of solutes change intracellular osmolality. The purpose of this study was to examine the role of Ca^{2+}, a ubiquitous intracellular messenger, in regulated volume decrease by *Necturus maculosus* erythrocytes. Osmotic fragility, examined with a spectrophotometer, increased in the presence of a 0 Ca^{2+}-EGTA Ringer, suggesting a role for Ca^{2+} in volume regulation. We next measured changes in cell volume in response to hypotonic (0.5X) amphibian Ringer with a Coulter counter. Buffering intracellular Ca^{2+} with BAPTA-AM (100 μM) reduced the percent volume decrease over a 120 minute period. In contrast, increasing intracellular Ca^{2+} with A23187 (0.5 μM, a Ca^{2+} specific ionophore) potentiated volume recovery. Further, gadolinium (10 μM, an antagonist of Ca^{2+} influx) and hexokinase (2.5 U/ml), which dephosphorylates ATP in the presence of glucose, both inhibited volume recovery. The effect of these agents were reversed with A23187 and gramicidin (2 μM, a cationophore used to maintain a high K^+ permeability). Consistent with the inhibitory action of hexokinase, the general P2 antagonist suramin (100 μM) blocked volume regulation. Conclusions: an increase in intracellular Ca^{2+} stimulated volume decrease by enhancing K^+ efflux in swollen cells. In contrast, volume regulation was inhibited by buffering intracellular Ca^{2+}. In addition, our results are consistent with a rise in intracellular Ca^{2+} being linked to extracellular ATP activation of a P2 receptor during cell swelling. (Funded by NSF grant MCB-0076006)